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Prognostic Factors For Functional Outcome in Patients with Mesencephalic Hemorrhage

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ABSTRACT

Introduction: Mesencephalic hemorrhage (MH) is a rare presentation of spontaneous intraparenchymal hemorrhage. This study aims to evaluate prognostic parameters of the MH outcome.

Methods: We conducted an extensive search in the literature for cases with spontaneous, isolated mesencephalic hemorrhage. The study was conducted according to the statement of Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA). Sixty-two eligible cases have been reported in the literature as proven by CT or MRI, and to these, we added six cases confirmed by MRI. The modified Rankin Scale (mRS) was dichotomized into two groups as the favorable outcome (FO; score, 0–2) and unfavorable outcome (UO; score, 3–6).

Results: Of the 68 patients studied, 26 (38%) presented with normal consciousness, 22 (32%) with lethargy , and 20 (29%) with stupor or coma. There was no cause of hemorrhage in 26 (65%) patients with FO and 12 (43%) with UO (p=0.059). In univariate analyses, neither arteriovenous malformations (p=0.33) nor cavernomas (p=0.19) were associated with outcome. Multiple logistic regression analysis revealed that hypertension (OR, 51.22; CI95%, 1.92–1370.24; P=0.019), consciousness (OR, 133.54; CI95%, 1.61–1113.3; P=0.03), NIHSS at admission (OR, 57.23; CI95%, 2.87–1141.2; p=0.008), and ventrodorsal hemorrhage size (≥1 cm) (OR, 61.83; CI95%, 2.15–1779.2; p=0.016) were significantly associated with UO. Three months after stroke, 40 patients (59%) had FO, 28 (41%) had UO, and 8 (12%) died.

Conclusion: These results suggest that ventrodorsal size of hemorrhage and clinical severity at stroke onset are possible predictors of functional outcome after mesencephalic hemorrhage.

Keywords: Mesencephalic hemorrhage, brainstem hemorrhage, predictive parameters, modified rankin scale

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INTRODUCTION

Primary spontaneous mesencephalic hemorrhages (MH) have been rarely reported. The clinicanatomical features are characteristic in comparison to pontine, medullary, and similar intraparenchymal hemorrhagic lesions in which some patients have a favorable outcome (FO) (1–8), while other patients have an unfavorable outcome (UO) and death (4,9–12).

There are definitive criteria to predict the neurological outcome of MH using clinical and neuroimaging parameters. Computer tomography (CT) scan is a common imaging method for evaluating patients with stroke. However, it has diagnostic limitations of precise information in the early hemorrhage period due to lack of precision caused by poor spatial resolution, difficulty distinguishing between new and old blood, and poor demonstration of edema. These limitations can be overcome with specific gradient recalled echo (GRE), and susceptibility-weighted imaging (SWI) magnetic resonance imaging (MRI) modalities.

Thus, in order to enlighten the predictive outcomes in MH patients we assessed the cases in the literature plus six additional neuroradiologically-confirmed patients.

Highlights

- **The ventrodorsal size of hemorrhage and clinical severity at stroke onset are predictors.**
- **There is no significant clinical symptom which can predict outcome of mesencephalic hemorrhage.**
- **Hypertension and state of consciousness at the time of mesencephalic hemorrhage may be associated with poor prognosis.**

METHODS

Our study subjects were recruited from cases reported in the literature and ongoing single-center prospective stroke records. Between January 1, 2014, to January 30, 2019, 3200 patients with stroke were enrolled and

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prospectively entered in our Stroke Registry. Among 382 (12%) patients with CT/MRI-proven intracerebral hemorrhage, six had a hematoma restricted to the mesencephalon while aneurysm and vascular malformations were excluded through CT angiography after a routine clinical assessment procedure for with a prospectively recording of all variables for the study. This study was approved by the ethical committee of Alaaddin Keykubat University with approval number E. 688414/15.5.22.

Magnetic Resonance Imaging Acquisition and Analysis

Magnetic resonance images were acquired on 1.5 and 3-Tesla Signa scanners (Siemens Sonata, Siemens Medical Solutions, Erlangen, Germany). Routine MRI sequences including DWI, fluid-attenuated inversion recovery (FLAIR), T1-weighted imaging, T2-weighted imaging, and susceptibility-weighted imaging and gradient recalled echo T2* were acquired as defined in our previous study in detail. We defined microhemorrhages on GRE and SWI sequences as hypointense-rounded lesions <10 mm in size irrespective of their location at surface or deeper location. Patients without CT and MRI results were not considered eligible for the study.

A stroke neurologist and 2 neuroradiologists blinded to each other's decisions, visually identified and sequentially outlined the lesion location and size on CT and MR images of all patients. To assess the maximum dimension of hemorrhage, the ventro-dorsal length of each hemorrhage was measured on axial CT and MRI while rostro-caudal size.

of each bleeding of previous reports was not measured due to failed data. The maximum dimension of hemorrhage was subdivided into two groups as small hemorrhage (<1 cm) and large hemorrhage (≥1 cm) for analysis based on the ventrodorsal length of the hemorrhages.

Search Strategy and Data Extraction of Reported Cases with Mesencephalic Hemorrhage

The report of this systematic review was made according to the recommendations of Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (www. prisma-statement. org) (Figure 1). Data written in English and French on hemorrhage in the mesencephalon (or midbrain) were sought from computerized databases (MEDLINE, Scopus, EMBASE, Science Citation Index) using terms and strategies based on the Cochrane Stroke Group methodology given in the supplementary section in detail.

Additional Methodological Details

To detect isolated "mesencephalic hemorrhage" or "midbrain hemorrhage" correctly, the authors scanned the reference lists of the included studies or relevant articles identified through the search. We included only the patients with CT and MRI results according to the study protocol. The researchers scanned the obtained titles and abstracts considering the inclusion criteria. They received full reports for all topics where they met the participation criteria or where there was any uncertainty. Authors screened the case reports or series, and decided whether these met the inclusion criteria. They resolved any disagreement through discussions. Neither of the authors were blind to the journal titles, manuscripts, or to the study authors or institutions. The following data were obtained from the studies included in the study: study authors, hemorrhage size, clinical features, outcome, and limitations. The methodological quality of the included studies was individually evaluated by the authors and consensus was finally reached.

The total number of patients eligible for analysis was 62 in the literature and 6 in our database (38 men and 30 women). We did not include studies without CT and MRI that did not allow us to assess the extent of hemorrhage. For the current analysis, baseline clinical variables (age, vascular risk factors, stroke severity, consciousness state, pupillary light reflex, diplopia, vertical gaze palsy, third and fourth cranial nerve palsy, motor and sensory deficits) and imaging (including the findings of CT/MRI, and CT/MR angiography) characteristics were recorded. The neurological severity of cases was assessed using the National Institutes of Health Stroke Scale (NIHSS) scores on admission, day 2 or 3 of hospitalization, and at discharge. Consciousness disorder was defined as impaired wakefulness and awareness such as drowsiness, stupor and coma. The outcome of the patients was assessed by examination at the outpatient service with stroke program seniors during discharge and 3 months after stroke. Therapy (surgery, radiosurgery) of patients was

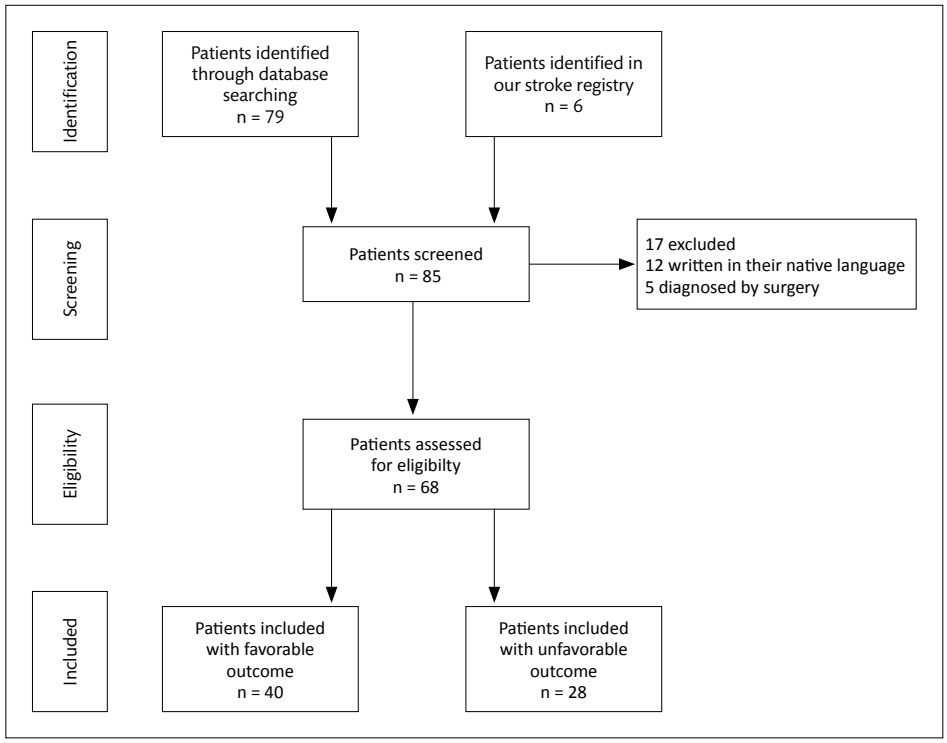

Figure 1. The flow of research through the different phases of the study regarding "preferred reporting items for systematic reviews and meta-analyses (PRISMA)".

Table 1. Mesencephalic hemorrhages with favorable outcome: demographic, clinical findings, etiology and outcome

Author/year Age/sex	Consciousness	Pupil	Ptosis	Eye movements	Other cranial nerves	Pyramidal signs	Sensory/ cerebellar signs	Etiology	Size of Hemorrhage (cm)	Outcome (mRS)
Our present case 1 65/M	Normal	Normal	-	Vertical gaze palsy, skew deviation	-	-	-	HT	<1	0
Our present case 2 59/M	Normal	Normal	-	Diplopia	Abn IVCN	-	-	Unk	<1	0
Our present case 3 67/M	Drowsiness	Midriasis, ALR (U)	U	Oculomotor palsy, (Weber's syndrome)	-	U	-	Amyloid angiopathy	≥1	2
Our present case 4 48/F	Normal	ALR (B)	B	Vertical gaze palsy	-	-	-	Amyloid angiopathy	<1	0
Our present case 5 39/F	Normal	Midriasis, ALR (B)	-	Oculomotor palsy, (Nothnagel's syndrome)	-	-	Gait ataxia	Cavernoma	<1	0
Our present case 6 42/M	Stuporous	Unequal, ALR	-	Impaired vertical eye movements	-	U	-	Cavernoma	<1	1
Humphreys et al., 1978 10/F	Coma	ALR	U	Oculomotor palsy	-	B	Ataxic	AVM*	≥1	2
La Torre et al., 1978 38/F	Drowsiness	Normal	-	Vertical gaze palsy	Abn VICN	B	-	AVM*	<1	1
Brismar et al., 1979 37/F	Drowsiness	Normal	B	Vertical and leftward palsy	-	-	Face hypoesthesia	Unk	<1	1
Zuccarrello et al., 1980 1/57/F 2/53/F	Drowsiness Normal	Normal Normal	U -	Vertical gaze palsy Oculomotor palsy	- -	U -	Gait ataxia -	Unk Unk	<1 <1	1 1
Morel-Maroger et al., 1982 71/M	Normal	Normal	-	Oculomotor palsy	-	U	Ataxic	HT	≥1	2
Tuttle & Reinmuth, 1984 52/M	Normal	Normal	-	-	Nystagmus	-	Limb numbness	HT	<1	1
Shuaib &Murphy, 1987 1/17/M 2/51/M	Normal Normal	Normal Unequal, ALR	U U	Diplopia, oculomotor palsy Oculomotor palsy	- -	- -	- -	Unk Unk	≥1 <1	2 1
Mehler & Ragon, 1988 1/28/M 2/36/M	Drowsiness Stuporous	Miosis, ALR ALR	B	Oculomotor palsy, CRN Vertical gaze palsy	Abn VICN	U U	Gait ataxia	Unk Unk	<1 ≥1	1 2
Tronci & Congia, 1989 35/M	Normal	Unequal, ALR	U	Oculomotor palsy, diplopia	-	U	Limb paresthesia	Unk	≥1	1
Han et al., 1989 1/40/M 2/38/M 3/39/M	Drowsiness Drowsiness -	- Unequal, ALR ALR	- U -	CRN Oculomotor palsy INO	- - -	- - -	Hemihypalgesia - Gait ataxia	Unk HT Unk	<1 <1 <1	1 0 1
Fingerote et al., 1990 1/35/F 2/30/F 3/56/F 4/51/M 5/17/M	Normal Drowsiness Drowsiness Normal Drowsiness	Unequal, ALR Unequal Unequal, ALR Unequal, ALR -	- - - U -	Oculomotor palsy, diplopia - CRN, diplopia, upward palsy Horizontal nystagmus Oculomotor palsy	- - - - -	- U U, asterixis,- - U	-- Limb paresthesia Gait ataxia Gait ataxia -	Unk Unk Unk Unk Unk *	<1 ≥1 <1 <1 ≥1	0 1 1 0 1
Link et al., 1993 1/55/M 2/44/F 3/71/M	Normal Drowsiness Confusion	Normal Normal Normal	- - -	- - -	- Abn IVCN Abn IVCN	U - -	- Hemihypalgesia Limb numbness	Unk Unk Unk	<1 <1 <1	2 0 1
Shintani et al., 1994 79/F	Normal	ALR	U	Oculomotor palsy	-	-	-	Unk	<1	0
Tomecek & Morgan, 1994 65/F	Normal	ALR	B	Oculomotor palsy	-	-	-	Unk	<1	1

Continuation of Table 1

Esteban Muñoz et al., 1996 46/F	Normal	ALR	-	Upward-downward palsy	Dysarthria	U	Hypoesthesia, ataxia	Unk	≥1	1
Lee et al.,1006 48/F	Normal	ALR	-	CRN, upward gaze palsy	-	-	-	Unk	<1	0
Işikay et al., 2000 73/M	Normal	Myosis	B	Oculomotor palsy	-	-	-	HT	<1	1
Rodríguez-Gómez et al., 2000 39/M	Normal	Normal	-	Upward-downward palsy	-	-	-	Unk	<1	0
Mizushima & Seki, 2002 75/M	Normal	Normal	-	Oculomotor palsy, diplopia	-	-	-	HT	<1	0
Bhola &Olson, 2006 43/M	Normal	Normal	-	CRN, diplopia	Abn IVCN	-	-	Unk	<1	0
Perez &Nunez, 2008 83/F	Normal	Normal	-	Vertical binocular diplopia	Dysarthria	-	Gait instability	Cavernoma	<1	1
Nguyen et al., 2017 14/M	Stuporous	ALR	-	Vertical gaze palsy, diplopia	-	U	-	HT**	<1	1
Turkes, 2017 42/F	Normal	Normal	-	Upward gaze palsy, skew deviation	-	-	-	Unk	<1	0

R: right; L:left; B: bilateral; U: unilateral; F: female; M: male; CRN; convergence retraction nystagmus; ALR: abnormal light reflex; AVM: arteriovenous malformation; HT: Hypertension; mRS: Modified Rankin Scale (3 months after stroke); Unk: unknown; Abn: Abnormal IVCN: Fourth cranial nerve; VICN: Sixth cranial nerve.
*hematoma evacuated.
**treated byendoscopic third ventriculostomy.

also recorded. The modified Rankin Scale (mRS) was used to measure the outcome of stroke and the degree of disability or dependence in the daily activities of patients. mRS score 0–2 was considered as a favorable outcome (FO), and mRS 3–6 score as the unfavorable outcome (UO).

RESULTS

Our study population consisted of 30 men and 38 women, aged 49.1±17.3 years (mean ± standard deviation (SD), median: 47 years). Demographic and clinical data of the patients are presented in Tables 1 and 2 in the Data Supplement.

With regard to consciousness level, 26 (65%) patients with FO and 2 (7%) with UO had normal consciousness at stroke onset (p=0.001). Seventeen patients (61%) who were in a coma at admission showed UO. Neurological symptoms determined by NIHSS score (>10) on the first day of stroke were higher in patients with UO (86%) than those with FO (23%) (OR, 15.82; CI95%, 4.47–56.09; P=0.001) (Table 3 in the Data Supplement). Nineteen (48%) patients with FO and 19 (68%) with UO had abnormal light reflex at admission (OR, 2.33; CI95%, 0.85–6.39; P=0.078). Partial or complete III. cranial nerve involvement was found in 15 (38%) patients with FO and 18(64%) with UO (p=0.027). Vertical gaze palsy was present in 12 (30%) patients with FO and 10 (36%) with UO (p=0.41). Patients with UO showed a significantly higher rate of larger ventrodorsal size (≥1 cm) than those with FO (OR, 28.70; CI95%, 7.02–117.44; p=0.001). Hypertension was significantly more frequent in patients with UO (39%) compared to those with FO (18%) (p=0.04). Conventional radiological examinations (CT and/or MRI) showed no cause of hemorrhage in 14 (35%) patients with FO and 16 (57%) with UO (p=0.059). Arteriovenous malformation (5 cases), cavernoma (3 cases), and coagulopathy (2 patients) were uncommon causes. Two of our patients had multiple micro-hemorrhages in different subcortical territories, including the bilateral caudate nucleus and putaminal regions. Our patients P5 and P6 had a hemosiderin ring and MRI signal characteristics of a recent hemorrhage in the midbrain suggesting small cavernoma malformation (Figure 2 in the Data Supplement.). Overall, there were eight patients with vascular malformations, including arteriovenous malformations and cavernomas, but none of them had any effect on the outcome (p=0.33 and p=0.19, respectively). Multiple logistic regression analysis showed that hypertension, NIHSS at admission, consciousness disorder at admission, and hemorrhage size (≥1 cm) were significantly associated with UO (R^2:0.83) (Table4 in the Data Supplement.).

Three months after stroke, five patients (10%) who had severe consciousness disturbance at stroke onset died. Forty patients (59%) had FO three months after hemorrhage, and 28 patients (41%) had UO.

DISCUSSION

In this study, we found that increased hemorrhage size, especially the ventrodorsal size, and higher initial NIHSS scores were associated with poor outcomes. Furthermore, there were no sensitive clinical indicators for FO and UO and the involvement of pyramidal and cranial nerve signs seemed to be associated with UO. It can be suggested that corticospinal tract involvement is more frequent in UO patients than in FO patients. In line with this, isolated, small (<1 cm) MH occurring in the tectum and tegmentum of the midbrain region has a FO as reported previously (10,11). Although the frequency of oculomotor nuclear or fascicular palsy was higher in the UO group, vertical gaze palsy suggesting the involvement of the rostral interstitial nucleus of the medial longitudinal fasciculus (ri-MLF) or the projection of the ri-MLF to the oculomotor complex did not statistically differ between the two groups and did not provide a predictive outcome.

Age, sex, and diabetes mellitus did not predict outcome in our analysis, which is in accordance with previous studies (4,12,13). However, hypertension was significantly associated with UO. There is no statistical difference between known and unknown causes of MH, which may

Table 2. Mesencephalic hemorrhages with unfavorable outcome: demographic, clinical findings, etiology and outcome

Author/year Age/sex	Consciousness	Pupil	Ptosis	Eye movements	Other cranial nerves	Pyramidal signs	Sensory signs	Etiology	Size of Hemorrhage (cm)	Outcome (mRS)
Scoville & Poppen, 1949 44/F	Stuporous	Unequal, ALR	B	Oculomotor palsy	ACR	U	-	HT	≥1	4
Dhopesh et al., 1980 1/61/F 2/66/F	Drowsiness Drowsiness	- Unequal	- -	Vertical gaze palsy Oculomotor palsy	- -	U U	- -	HT HT	≥1 ≥1	3 3
Borregoet al., 1981 66/F	Coma	Unequal	-	Vertical gaze palsy, convergence spasm	-	B	Ataxic	HT	≥1	6
Roig et al., 1982 1/56/F 2/56/F 3/62/M	Normal Drowsiness Coma	- Unequal, ALR ALR	- U -	Oculomotor palsy Oculomotor palsy AOC, AVR	- - ACR	- - B	- - -	HT HT -	<1 ≥1 ≥1	3 5 6
Durward et al., 1982 18/M	Stuporous	-	-	Oculomotor palsy	Abn IVCN	U	-	AVM*	≥1	4
Sand et al., 1986 1/42/M 2/37/M 3/54/F	Normal Drowsiness Drowsiness	- Miosis, ALR Miosis	- B U	Vertical gaze palsy, CRN Vertical gaze palsy, hypotropia Abn IVCN	Dysarthria Hyperacusis	- B -	Lid paresthesia Gait ataxia Gait ataxia	Unk Unk Unk	≥1 ≥1 ≥1	3 4 3
Weisberg et al., 1986 1/39/M 2/44/F 3/45/M 4/36/F 5/52/M 6/48/M	Drowsiness Drowsiness Drowsiness Drowsiness Stuporous Stuporous	Midriasis, ALR Midriasis, ALR Unequal, ALR ALR ALR Miosis	U U - - - -	Vertical gaze palsy Vertical gaze palsy Vertical gaze palsy Oculomotor palsy Vertical gaze palsy Oculomotor palsy	Abn VICN - - - - -	B U - U U U	- Ataxic (U) - - - -	Unk Unk Unk Unk Unk Unk	≥1 ≥1 <1 ≥1 <1 ≥1	4 3 3 3 3 3
Keane et al.1988 38/M	Coma	ALR	U	Oculomotor palsy	-	-	-	Unk	≥1	3
Mehler & Ragon, 1988 36/M	Stuporous	Miosis, ALR	B	Oculomotor palsy, vertical gaze palsy	Abn VICN	U	Limb paresthesia	HT	≥1	4
Mendonça et al., 1990 70/F	Stuporous	Midriasis (B)	B	Oculomotor palsy	OC+VOR (-)	B	-	HT	≥1	6
Link et al., 1993 1/75/M 2/51/F 3/81/M 4/30/F.	Coma Stuporous Stuporous Stuporous	Midriasis, ALR ALR (B) ALR (B) ALR(B)	B B B B	Oculomotor palsy Oculomotor palsy Oculomotor palsy (B) Oculomotor palsy (B)	Abn VICN Dysarthria Anarthria Dysarthria	B U U U	- Limb paresthesia - Gait ataxia	HT, WF AVM** Heparin AVM*	≥1 ≥1 ≥1 ≥1	6 3 6 4
Raison et al., 2008 1/70/M 2/91/F 3/42/M	Coma Stuporous Coma	Midriasis, ALR Unequal, ALR Midriasis, ALR	B U B	Oculomotor palsy (B) - Oculomotor palsy (B)	Anarthria Abn VICN Anarthria	B U B	- Gait ataxia -	HT Unk HT	≥1 ≥1 ≥1	6 4 6
Lee et al., 2018 39/M	Stuporous	Unequal, ALR	U	Oculomotor palsy (B)	Dysarthria	B	Gait ataxia	Unk	≥1	6

R: right; L: left; B: bilateral; U: unilateral; Abn: Abnormal; IVCN: Fourth cranial nerve; VICN: Sixth cranial nerve; ACR: abnormal corneal reflex; ALR: abnormal light reflex; AOC: abnormal oculucephaic reflex; AVR: abnormal vestibulo-ocular reflex; CRN; convergence retraction nystagmus; HT: Hypertension; mRS: Modified Rankin Scale; OC+VOR (-): abolition of oculocephalic and vestibuloocular reflexes; WF: warfarin; Unk: unknown. *treated by shunt; **treated by gamma knife irradiation.

Table 3. Univariate analysis of predictors for outcome

	Favorable Outcome N= 40	Unfavorable Outcome N= 28	Univariate OR (95% CI)	P-value
Age (age+SD)*	47.2+17.8	51.8+16.6		0.29
Sex (Men)	17 (43)	13 (46)	1.17 (0.44-3.10)	0.47
Hypertension	7 (18)	11 (39)	3.05 (1.0-9.29)	0.04
Diabetes mellitus	4 (10)	4 (14)	1.50 (0.34-6.58)	0.43
Consciousness disorder at admission	14 (35)	26 (93)	24.14 (4.98-116.99)	0.001
NIHSS at admission (Score ≥10)	11 (28)	24 (86)	15.82 (4.46-56.09)	0.001
Cause of hemorrhage AVM Cavernoma Amyloid angiopathy	 2 (5) 3 (8) 2 (5)	 3 (11) 0 0	 2.28 (0.36-14.63)	 0.33 0.19 0.34
Hemorrhage size (≥1cm)	9 (23)	25 (89)	28.70 (7.02-117.44)	0.001
Oculomotor nerve involvement	15 (38)	18 (64)	3.0 (1.10-8.19)	0.027
Cranial nerves involvement including VICN, IVCN	9 (23)	17 (61)	5.32 (1.84-15.38)	0.002
Pyramidal signs	15 (38)	22 (79)	6.11 (2.02-18.48)	0.001

IVCN: Fourth cranial nerve; VICN: Sixth cranial nerve
*ANOVA test, SD: standard deviation
**Values in parentheses are percentage of column.
AVM: arteriovenous malformation; NIHSS: National Institutes of Health Stroke Scale.

predict the clinical outcome in MH. Here, it is reasonable to assume that some previous unknown cases can be detected with either new CT or MRI techniques, such as thrombosed malformations, cavernomas, and bleeding due to cerebral amyloid angiopathy (CAA). Notably, we identified multiple micro-hemorrhages in different subcortical territories –including bilateral caudate nucleus and putamen– of two patients with GRE and SWI, which has been shown to be strongly associated with CAA (5). However, it is worth mentioning here that our study is limited to a few cases with multiple microhemorrhages. Therefore, further studies are needed to clarify the role of CAA in MH.

The study has some minor limitations. First, there was no uniformity in evaluation of the lesion size due to the increasing accuracy of imaging techniques during the recruitment period. In line with this, only the ventrodorsal plane size was available on published reports, and multi-directional measurement approaches, such as the ventrodorsal and rostrocaudal axis are needed for an accurate clinical evaluation. For instance, a rostrocaudal direction of the hemorrhage may be associated with a more severe clinical picture due to pyramidal tract involvement. Second, the follow-up period is relatively short, consisting of rare case reports.

Due to the rare cases of mesencephalic hemorrhage in the literature, the study's major strength is that it provides strong predictive clinical data in the early hemorrhage period of patients with midbrain hemorrhage through novel hemorrhage-sensitive MRI imaging modalities.

Conclusion

In conclusion, the ventrodorsal size of hemorrhage and clinical severity at stroke onset are possible predictors of the outcome of MH. New generation CT scanners and MRI sequences with sharper and high-quality images will help to visualize the lesion and clarify the underlying pathophysiology by giving a piece of meaningful diagnostic information.

Table 4. Multiple logistic regression analysis of predictors for unfavorable outcome

	Adjusted OR	(95% CI)	*p*-value
Hypertension	51.22	1.92-1370.24	0.019
Consciousness disorder at admission	133.54	1.61-1113.3	0.03
NIHSS at admission (Score ≥10)	57.23	2.87-1141.2	0.008
Hemorrhage size (≥1cm)	61.83	2.15-1779.2	0.016
Cranial nerves involvement	7.92	0.57-110.73	0.12
Pyramidal sign	0.02	0.001-2.34	0.11

NIHSS: National Institutes of Health Stroke Scale; OR, odds ratio.
Overall percentage in classification table was 91.2
Nagelkerke R^2: 0.83
Hosmer-Lemeshow goodness-of-fit: p=0.73

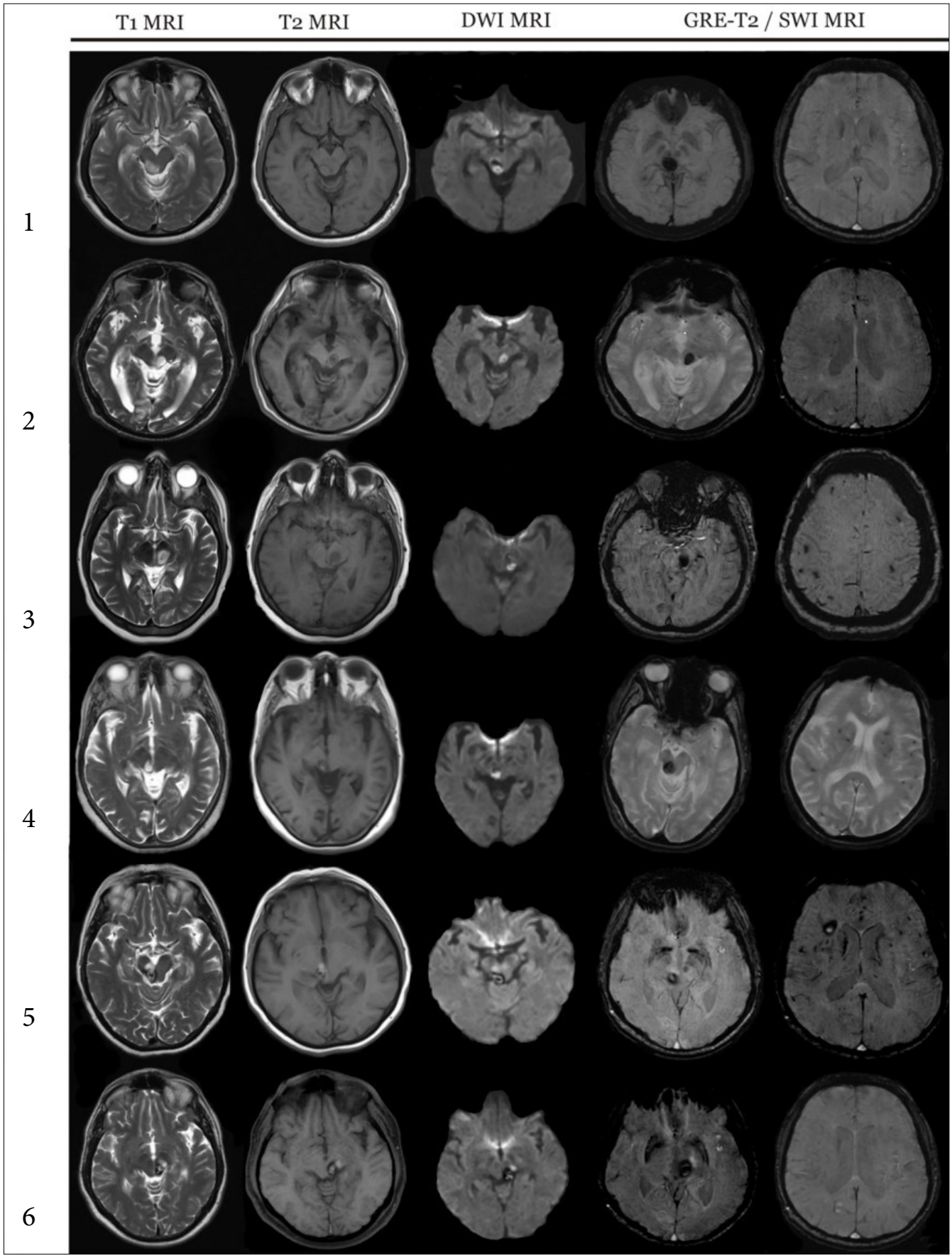

Figure 2. MRI sequences of the present series demonstratemesencephalic hemorrhage in 6 patients. Susceptibility-weighted imaging (SWI) and T2*-weighted gradient-recalled echo (GRE) identified cerebral microhemorrhages (axial plane) in the parietal lobe and deep white matter in patients P3 and P4. Patients P5 and P6 had a rim of signal loss due to hemosiderin and MRI signal characteristics of a recent hemorrhage in the midbrain suggesting small cavernoma malformation. Patient P5 had small cavernomas with hemorrhagic component in the temporal and parietal subcortical areas which were demonstrated by SWI MRI.

(*) These authors contributed equally to this work.

Ethics Committee Aproval: This study was approved by the ethical committee of Alaaddin Keykubat University with approval number E. 688414/15.5.22.

Informed Consent: Informed consent was obtained from all participants.

Peer-review: Externally peer-reviewed

Author Contributions: Concept– EK, FEB; Design– EK, FEB; Supervision– BY, EÖÖ, SÇ, AÖ; Resources– (-); Materials– (-); Data Collection and/or Processing– EK, MMD, FEB, EÖÖ; Analysis and/or Interpretation– EÖÖ, EK, FEB, AÖ, MMD, ŞÇ, BY, WRS; Literature Search– MMD, EK; Writing Manuscript– EÖÖ, BY, EK; Critical Review– EÖÖ, BY, EK.

Conflict of Interest: The authors declared that there is no conflict of interest.

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